1. Introduction

The sinus node is the origin of cardiac activity and generates the contraction commands but the mechanism by which a sinus rhythm is determined, is not fully understood [1]. It has been admitted especially by physicians, that normally a cell having the highest frequency drives the other cells [2–4]. However, Dehaan and Hirakow [5] reported that on the contact of pairs of spontaneously beating myocytes of chicken hearts, some were synchronized at a higher frequency than that of the higher member, some at a frequency lower than the lower member, and the others at an intermediate frequency of the two. Sano et al. [6] found an inter-neuronal electronic influence on their firing rates in the sinus node of a rabbit and obtained the phase response curve (PRC).

These results, suggest that the synchronization among pacemaker neurons plays a crucial role in the determination of the sinus rhythm and that the mechanism appears to be complex. Electrophysiological studies have suggested that the activity of cardiac cells with automaticity (e.g. in the Sino-Atrial (SA) node, Purkinje network, Atrial and Ventricular myocardium) can be modulated by stimulating current pulses (sub-threshold depolarizing or hyperpolarizing) applied extra-cellularly [6–8]. The effects of external stimuli on the frequency of biological oscillators were observed in a wide range of species, and their overall characteristics can be well described by a PRC [7–9]. Systematic perturbation techniques (by stimulating the pacemaker at various phases of its intrinsic cycle) have been applied to experimentally investigate and to establish the shape of the PRC [10,11].

Numerous modeling has been performed on cardiac cells and electric propagation based on PRC until now. Wei et al. [12] modeled cardiac in three dimensional form based on the PRC and simulated supraventricular tachycardias and the Wolff–Parkinson–White (WPW) syndrome. In [13], a cardiac computer model is used with M cells and three dimensional excitation sequences during torsade de point are detected. The initiation, propagation and termination of that arrhythmia are also simulated in [13]. The effects of automatic Vagal and sympathetic nervous system were investigated by Abramovitch and Akselrod in 1998 [14] based on the PRC. Moreover, the effects of a train of pulses on pacemakers were studied and the entrainment regions of the pacemaker with input pulses by the ratio \( m:n \) were obtained also. In [15], atrium has